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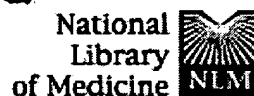
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Fatal lymphoreticular disease in the scurfy (sf) mouse requires T cells that mature in a sf thymic environment: potential model for thymic education.

Proc Natl Acad Sci U S A. 1991 Jul 1;88(13):5528-32.
PMID: 2062835 [PubMed - indexed for MEDLINE]

☐ 2: [Lyon MF, Peters J, Glenister PH, Ball S, Wright E.](#) Related Articles, Links

The scurfy mouse mutant has previously unrecognized hematological abnormalities and resembles Wiskott-Aldrich syndrome.

Proc Natl Acad Sci U S A. 1990 Apr;87(7):2433-7.
PMID: 2320565 [PubMed - indexed for MEDLINE]

☐ 3: [Patel DD.](#) Related Articles, Links

Escape from tolerance in the human X-linked autoimmunity-allergic dysregulation syndrome and the Scurfy mouse.

J Clin Invest. 2001 Jan;107(2):155-7. No abstract available.
PMID: 11160129 [PubMed - indexed for MEDLINE]

☐ 4: [Clark LB, Appleby MW, Brunkow ME, Wilkinson JE, Ziegler SF, Ramsdell F.](#) Related Articles, Links

Cellular and molecular characterization of the scurfy mouse mutant.

J Immunol. 1999 Mar 1;162(5):2546-54.
PMID: 10072494 [PubMed - indexed for MEDLINE]

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The C. elegans PTEN homolog, DAF-18, acts in the insulin receptor-like metabolic signaling pathway.

Ogg S, Ruvkun G

Department of Molecular Biology, Massachusetts General Hospital, Boston 02114, USA.

An insulin-like signaling pathway, from the DAF-2 receptor, the AGE-1 phosphoinositide 3-kinase, and the AKT-1/AKT-2 serine/threonine kinases to the DAF-16 Fork head transcription factor, regulates the metabolism, development, and life span of *Caenorhabditis elegans*. Inhibition of daf-18 gene activity bypasses the normal requirement for AGE-1 and partially bypasses the need for DAF-2 signaling. The suppression of age-1 mutations by a daf-18 mutation depends on AKT-1/AKT-2 signaling, showing that DAF-18 acts between AGE-1 and the AKT input to DAF-16 transcriptional regulation. daf-18 encodes a homolog of the human tumor suppressor PTEN (MMAC1/TEP1), which has 3-phosphatase activity toward phosphatidylinositol 3,4,5-trisphosphate (PIP3). DAF-18 PTEN may normally limit AKT-1 and AKT-2 activation by decreasing PIP3 levels. The action of daf-18 in this metabolic control pathway suggests that mammalian PTEN may modulate insulin signaling and may be variant in diabetic pedigrees.

PMID: 9885576, UI: 99102962

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